PHILADELPHIA NEUROLOGICAL SOCIETY.

December 17, 1900.

The President, Dr. William G. Spiller, in the chair.

Dr. D. J. McCarthy presented a case of amyotrophic lateral sclerosis, sent to the nervous clinic of the Polyclinic Hospital by Dr. S. Solis Cohen, in which the atrophy and paralysis were almost confined to the muscles of the shoulders and upper arms, and the movements of the hands were good.

Dr. Francis X. Dercum said that he had seen the case on one occasion and regarded it as one of muscular atrophy of spinal origin. The rapid progress of the case he regarded as very unusual.

Dr. William G. Spiller said that syringomyelia sometimes caused this form of muscular atrophy and paralysis, even without the dissociation of sensation, but such cases were very rare.

Drs. C. K. Mills and T. H. Weisenberg presented cases illustrating the differential diagnosis of cerebral and hysterical hemianesthesia.

Dr. William G. Spiller said that many anatomists still hold the view of Charcot that a sensory tract exists in the posterior part of the posterior limb of the internal capsule, although they are unable to demonstrate the continuation of this tract in lower levels of the cerebro-spinal axis. Dejerine teaches that the fibers in the retrolenticular portion of the internal capsule come from the temporal lobe and that the thalamus and not merely the internal capsule must be implicated in these cases of persistent hemianesthesia. The lemniscus is probably a sensory tract, but there may be other sensory tracts. There is no evidence to show that the lemniscus is in relation with the retrolenticular portion of the internal capsule.

Dr. Spiller said that in two or three cases in which the Gasserian ganglion had been removed for tic douloureux he had found anesthesia on the operated side sharply limited by the median line of the face. In one of these cases the ganglion had been removed two or three years previously. Limitation of anesthesia at the median line of the body occurs in organic as well as hysterical anesthesia.

Dr. Charles K. Mills was not convinced of the truth of Dejerine's views that there is a complete admixture of fibers of sensation and motion in the limb of the capsule. Cases are on record which clearly indicate that from a one-sided lesion between the thalamus and cortex, in regions which are not motor in the ordinary sense of motor localization, there may be distinct disorders of sensation. Partial anesthesia has been present from lesion of the gyrus fornicatus. He had reported a case in which irregular anesthesia was associated with a lesion involving the cortex and subcortex of both parietal lobes. Cerebral lesions outside of the thalamus and outside of the motor region may give rise to anesthesias of various sort.

Dr. William G. Spiller said that he did not wish to be misunderstood. He had not discussed the diagnosis between thalamic and cortical lesions, but referred to the view that lesions of the posterior part of the posterior limb of the internal capsule cause anesthesia when the thalamus is not involved. It is very probable that cortical lesions may produce disturbance of sensation.

Dr. Charles K. Mills believed that instead of the sensory and motor fibers intermingling throughout the entire posterior limb of the capsule, the former were more or less concentrated posteriorly. He believed that there were two relays to the motor cortex. He held that while the thalamus was a great center for common sensibility a special system of neurones passed to the sensory cortex, and another from this region to the motor cortex.

Dr. Wm. Pickett read a paper on the insanities of adolescence.

Dr. Charles K. Mills considered the paper to be one of great value especially as presenting the results of the personal observation of the author.

Dr. Francis X. Dercum, referring to the fact that paranoia occurs at a later age than the other varieties, said that it was suggestive that the later the degeneration begins the more is it like that of middle life.

The katatonic cases are interesting because they afford a rather more hopeful prognosis than the other varieties. He had seen a limited number of cases of mild hebephrenia also in which the mental degeneration fell to a certain level and then under powerful stimulation of nutrition and re-education surprisingly good results had been obtained. With regard to diagnosis, it is not an uncommon error to find that the diagnosis of melancholia has been made when subsequent observation shows the case to be one of hebephrenia.

Dr. Daniel E. Hughes said that the insanities of adolescence were increasing. He considered heredity one of the striking features in the diagnosis. There is a large group of insanities that develop during the ages of puberty and adolescence that are curable. In the period of life between the ages of 12 and 30 years we have acute manias and melancholias which run the ordinary course and recover, and in which no evidence of mental weakness remains, but in the group of cases that has been pictured by Dr. Pickett the tendency is to dementia. These insanities occur at a developmental time when the experiences of youth are becoming memories and these memories are organising into ideas and percepts. Whether the symptoms are those of the hebephrenic, katatonic or paranoiac type will depend upon the age at which they appear, the older in the adolescent period the more systematized the so-called delusions. It is well known that a fair proportion of the insanities peculiar to adolescence do make at least a temporary recovery. He had seen cases which after running the various cycles of mania and melancholia for two or three years gradually get well and go out into the world and remain for three or four years, and then return with the whole group of symptoms as they were originally, with a more rapid progress towards dementia. This brings up the question, Are these people who have this unfortunate heredity, properly cared for? Is there not something in their environment which has a tendency to cause this breakdown? Is it not the duty of some one to take hold of these youths and surround them with influences which may prevent them from becoming chronic dements?

Dr. C. W. Burr reported several cases in which astereognosis was a prominent symptom.

Dr. Francis X. Dercum said that he had recently gone over a number of cases with regard to this symptom to determine if it were possible to give it a definite clinical value. It may be present in peripheral nerve affections with sensory disturbance; it may be present in certain forms of cord disease as locomotor ataxia and ataxia papaplegia, and in other cases it is due to cortical involvement. In these the separate senses may be preserved, the patient recognizing weight, pain and pressure, but being unable to combine these sensations to form a concept. Such cases as these he thought must be referred to the cortex. Whether the superior parietal lobule is the only area lesion of which gives rise to astereognosis was doubtful, and future studies alone would prove this. At any rate he had found that preservation of the muscle sense was not essential, but loss of the spacing sense had almost invariably proven a serious defect.

Dr. Joseph Sailer said that he had been interested in one point to which he had seen no reference, that was the occurrence of transient astereognosis due to cerebral lesion, which he though comparable to the transient aphasia. He agreed with Dr. Burr that it is difficult to say which of the senses is most important in stereognosis. Experiments on normal persons led him to believe that the muscle sense is not of vital importance. He thought that considerable light would be thrown upon the subject by a series of studies made on normal individuals.

Dr. William G. Spiller had understood Dr. Burr to refer to his second case as "object blind," using the term in the same sense as "mind blind." This he considered an interesting observation as the lesion was only on one side of the brain in the angular and supramarginal gyri.

With regard to the statement that the center of stereognosis is in the posterior part of the superior parietal lobe, that is very different from saying that lesion of that part will cause astereognosis. If the former statement were correct then only those forms of sensation which are represented in the parietal lobe would have anything to do with stereognosis. We have reason to believe that the parietal lobe is concerned in sensation, but it is probable that the motor area is also partly sensory.

Dr. Charles K. Mills thought that the facts presented by Dr. Burr helped to substantiate the statements which he had made in regard to the old views of Charcot and the new views of Dejerine. He did not believe that either view represented the exact truth. He thought that it was a mistake to restrict the sensory area of the cortex to the superior parietal lobule. He believed that the sensory region comprised a large area surrounding the motor area on all sides, and abundant facts tend to confirm this view. A unilateral cerebral lesion probably produces complete anesthesia only when it is thalamic. He thought that Dr. Burr's patient had an organic lesion which was responsible for the sensory symptoms, and that there was large involvement of her cortical sensory areas with little or no destruction of her motor cortical areas. It may be that a partial thalamic lesion or a subcortical parietal lesion might explain the case.

Dr. Charles W. Burr said he believed there is a distinct area of the brain which we may call the tactile perceptive area in which are stored the tactile memories and in which are grouped together all the sensory factors which go to make up a concept of the thing handled. In order to get a true conception of an object this tactile concept is combined with visual and, in less degree or at least less frequently, with auditory, olfactory and gastatory concepts. He believed this tactile perceptive area to be in the hinder portion of the parietal lobe and entirely separate from the motor area. Stereognosis may be due to an absence of some of the primary sensory factors, or it may be caused by disease of the tactile perceptive area, in which case there is either a tactile amnesia or an inability to group together the different sensations, just as a patient may see objects and not conceive them, or may feel objects and not conceive them.

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NEURASTHENIA, ETC., WITH ESPECIAL INVOLVEMENT OF THE SENSORY NEURONE.

Dr. F. Savary Pearce reported the following case: The patient, a physician, aged 38, is a member of a family of nine, without nervous heredity. In his early life the man suffered extremely from torpid liver and constipation, and has always been compelled to use cathartics. He was in good health until four years ago. Moving from the country to a city and to work largely of a clerical nature which confined him indoors for long hours, he soon developed trouble from the liver again, accompanied by vague feelings in his head as of a band pulled about the brow. His sleep became disturbed by what he thinks was intense paresthesia and causalgia in the feet. This annoying symptom extended up to the knees, although there was no anesthesia. He soon began to complain of numbness in the left arm, worse upon exertion and after a hearty meal. Vigorous massage would always alleviate this latter symptom. The next phenomenon was a sense of titillation in the larynx accompanied by frequent dry coughing. He noticed vague urinary symptoms as of "pain passing down the ureter." At this time, he particularly remarked the tendency to "catching-cold" easily. He was placed on aperients with copious draughts of hot water, and the use of digestants. His nervous symptoms improved under this treatment but the laryngeal irritation continued, although digestion and assimilation were never perfectly performed.

In July, 1899, the patient began to suffer from fleeting spasmodic pains in various muscles of the lower limbs. These attacks would occur at intervals of a few days to a few weeks, would seldom be localized at more than one point at a time, and would last for several hours or the entire day. Six months later the left skin surface of the upper lip became numb, and has continued so in varying degree until the present time, and the numbness is now symmetrical, embracing the entire chin and cheeks up to the lower eyelids. In addition to the feeling of numbness and actual "deadness" to all sense of pain, there is marked feeling of tension. A similar area of altered sensation at one time existed in the perineum. There is a general paresthesia about both gluteal regions, but down the outer sides of thighs there seems to be slight hyperesthesia. The knee-jerks are absent, but Romberg's sign or other evidence of organic disease are not present. Frequently during the waking hours, a sensation is experienced in the face as though five or six small shot or the ends of the same number of stiff hairs were thrust quickly against the skin. Sexual power is very much reduced. The stereognostic sense is normal. Examination of the urine has revealed marked indican reaction in several specimens, but never albumin or sugar. There has always been a marked difference between the morning and evening specific gravity, also irregular acid excretion, which may indicate alteration of general metabolism. The eye examination is entirely negative. Dr. Pearce believed that the disease was entirely one of neurasthenia with hypochondriacal manifestations; and that under nutritional measures, and change of scene, the sensory tract function would be restored.

Dr. Charles W. Burr suggested that the case might be hysterical. The analgesia changed from day to day. The laryngeal irritation would confirm with this view. Neither anesthesia nor laryngeal spasm is a symptom of neurasthenia.

Dr. Charles K. Mills spoke of the probability of the case being one of tabes. The loss of the Achilles-jerk, the diminution of the sexual power, and the coming and going cramps pointed in this direction, while the laryngeal attacks might be laryngeal crises.

Dr. H. A. Hare suggested that the case might as likely be one of pseudo-tabes due to neuritis. Many cases of hysteria in males are neurasthenic in origin. He had seen men utterly worked out, with loss of knee-jerk and less acuity of sensation than normal in certain portions of the body. He was inclined to think that the hysterical symptoms were due to nervous exhaustion. The crucial point in a case of this kind is the eye examination, which he understood had not yet been made. The eye examination will probably give useful information whether the case be hysterical or tabetic.

Dr. Wharton Sinkler had examined this patient six weeks ago and was satisfied that he was suffering from neurasthenia and hysteria. At that time the knee-jerks were absent but could be developed by reinforcement. He thought that the analgesia and the paresthesia were hysterical and that there was hysteria superimposed upon the neurasthenic condition.

Dr. F. S. Pearce considered that the neurasthenia was the fundamental condition. The man has been overworked and had every reason why he should suffer from neurasthenia. He considered the disease to be a so-called functional one, and that under appropriate hygiene and treatment the mental depression of the patient would be removed first, then physical restoration to health would follow. The absence of knee-jerk will occasionally be found in such cases of malnutrition of the central nervous system.